CHRONIC DUODENAL STASIS*

A SYNDROME WITH NEUROLOGICAL SYMPTOMS

By FREDERICK LEET REICHERT, M.D.
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DISCUSSION *by Nelson J. Howard, M.D., San Francisco; John Homer Woolsey, M.D., Woodland.*

THE clinical entity of chronic duodenal stasis was evolved some thirty years ago from an awakened interest in the seriousness of acute gastric dilatation. At that time, the reports of careful postmortem examinations witnessed by alert surgeons reëmphasized a pathological condition that had been mentioned sporadically in the literature since von Rokitansky,[1] in 1863, clearly described the acute gastric and duodenal dilatation from pressure of the mesenteric root on the third portion of the duodenum.

DISCUSSIONS IN THE LITERATURE

In Glenard's [2] classical presentation of enteroptosis, in 1889, a constriction in the duodenal-jejunal region is described as being caused by the traction produced by an acutely dilated stomach.

Fagge's [3] report, in 1873, on acute dilatation of the stomach, called attention to the relief experienced by the systematic use of the stomach tube, and the washing out of the gastric cavity with Vichy water, as suggested by Kussmaul [4] in 1869.

Schnitzler,[5] in 1895, in a case of acute gastric dilatation, believed due to mesenteric obstruction, reported a successful outcome by changing the patient from the dorsal to a ventral position.

Both Glenard [2] and Kundrat [6] felt that persistent, uncomplicated obstruction of the duodenum by the root of the mesentery was not uncommon, and that it led to gradual dilatation of the stomach and duodenum.

The mesenteric traction producing duodenal occlusion was thought to be due to the displacement of collapsed intestines into the pelvis. By this displacement, tension was put on the mesentery, thereby changing the cord-like superior mesenteric vessels into a constricting band that obliterated the lumen of the duodenum (Figs. 1 and 2). Albrecht's [7] paper, in 1899, strongly supported this theory.

Thomson,[8] in 1901 and 1902, discussed the different theories and favored the belief that, in a majority of the cases, a primary paralysis of the stomach was the underlying cause since, in his examinations, he found that the constrictions of the duodenum occurred at various levels, which would not be expected if the obstruction were due to compression by the superior mesenteric vessels.

Thomson's neuromuscular theory, of acute gastric dilatation, found further support in Laffer's [9] paper, in 1908, in which his experimental, clinical and pathological evidence indicated a primary disturbance of innervation affecting the gastric nerves, or their centers in the brain or cord. The recent

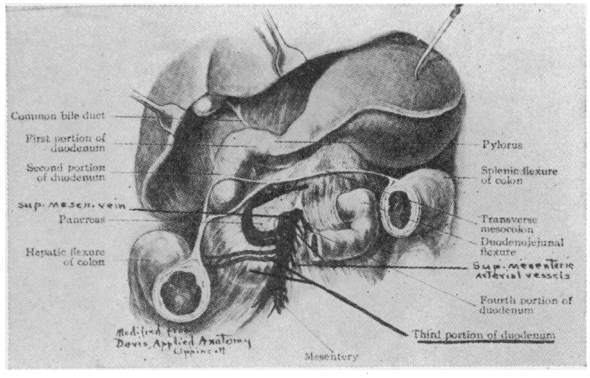

Fig. 1.—The duodenum, showing its course and relation to the surrounding organs. Compression of the third portion of the duodenum by the superior mesenteric vessels.

experimental work of Dragstedt, Montgomery, Ellis, and Matthews,[10] in 1931, again revealed that acute gastric dilatation was due to reflex inhibition of the peripheral motor mechanism through efferent impulses reaching the stomach by way of the vagi and splanchnics. They have postulated that stimulation of either visceral or somatic sensory nerves might produce such reflex gastric inhibition, and attributed the cause of death to failure to resorb the gastric and pancreatic juices and the inorganic elements, especially sodium and chlorin. The failure to resorb was dependent upon the inability of the atonic stomach and duodenum to propel the secretions into the lower intestines. In certain cases the occurrence of a secondary mesenteric obstruction to the inferior horizontal portion of the duodenum provided a further obstacle to the passage of secretions.

At the time of publication of Zade's [11] article, in 1905, on postoperative acute gastric dilatation or gastromesenteric ileus, as he termed it, the American surgeons were becoming aware of the seriousness of acute dilatation of the stomach, with Ochsner,[12] Finney,[13] and Bloodgood [14] reporting their observations. Finney felt that acute dilatation of the stomach and gastromesenteric ileus could not be differentiated; but Lewellys F. Barker,[15] in his discussion of Finney's reports, in 1905, advanced a differential diagnosis by the examination of the gastric contents for pancreatic ferments. It was also his suggestion that duodenojejunostomy be substituted for gastro-enterostomy, which had been found unsatisfactory.

Chronicity of this condition with clinically acute exacerbations was described by Bloodgood [16] in 1907, when he also proposed duodenojejunostomy as the proper operative procedure. The first duodenojejunostomy for chronic gastromesenteric ileus was performed by Stavely [17] in 1907, and reported by him, in 1908, as having been successful (Fig. 3).

Bloodgood in 1907, Stavely and Codman [18] in 1908, described and emphasized the chronic and more common condition of obstruction of the duodenum, and it is this time period which marks the recognition of a clinical entity termed chronic gastromesenteric ileus, chronic duodenal stenosis, chronic duodenal stasis or ileus, chronic intermittent duodenal obstruction and unstable or irritable duodenum.

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Read before the General Surgery Section of the California Medical Association at the sixty-sixth annual session, Del Monte, May 2–6, 1937.

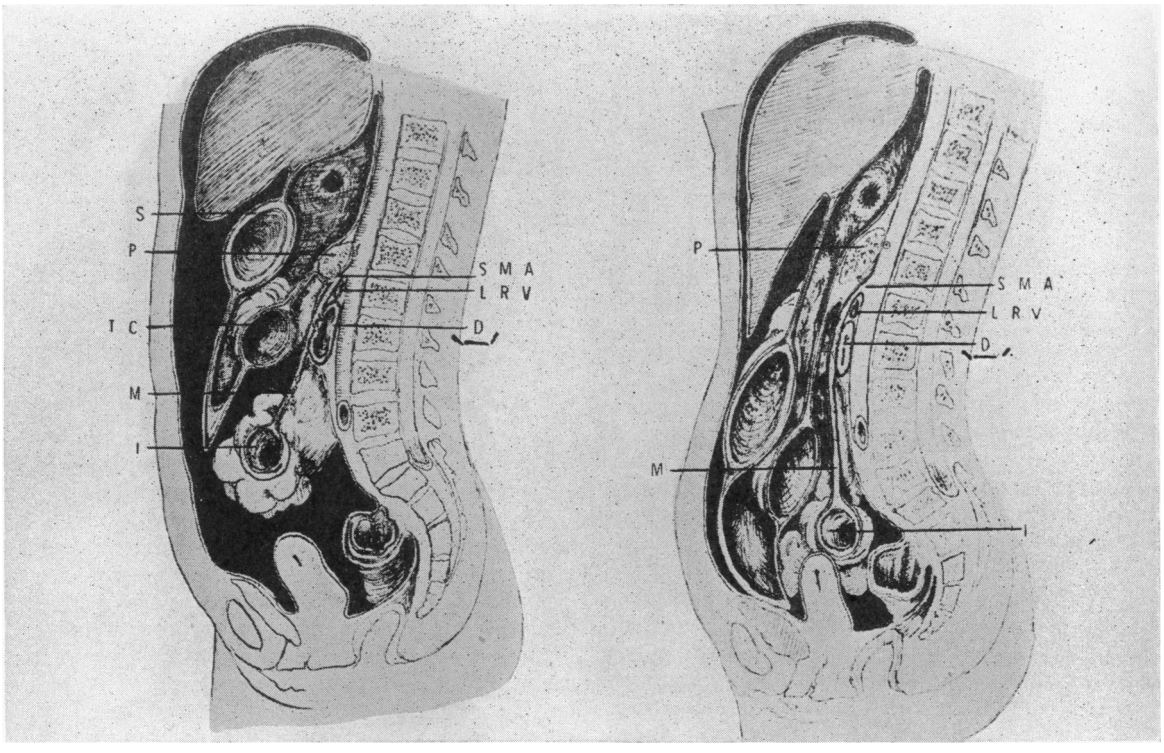

Fig. 2a Fig. 2b

Fig. 2a.—Transverse section of the normal duodenum (modified from Gray's Anatomy). S., stomach; P., pancreas; T.C., transverse colon; M., mesentery; I., ileum; S.M.A., superior mesenteric artery; L.R.V., left renal vein; D., duodenum.

Fig. 2b.—Drawing showing compression of the duodenum in the enteroptotic female. P., pancreas; M., mesentery; I., ileum; S.M.A., superior mesenteric artery; L.R.V., left renal vein; D., duodenum.

From J. McKenty: Chronic Duodenal Stenosis. (Permission for reproduction obtained from S. G. & O., and from author.)

Since 1908 the subject of chronic duodenal stasis has been kept before the medical profession, but little has been added to the earlier observations of symptoms and treatment. Papers by such authors as Wilkie,[19] McKenty,[20] Higgins,[21] Shattuck and Imboden,[22] and Friedenwald and Feldman,[23] are worthy of study.

AUTHOR'S PRESENTATION

My purpose in again presenting this subject is to emphasize the fact that certain symptoms may be so prominent that their cause is sought for elsewhere than in the toxicity of obstructed duodenal contents. I refer to those cases of chronic duodenal stasis with symptoms suggesting migraine,

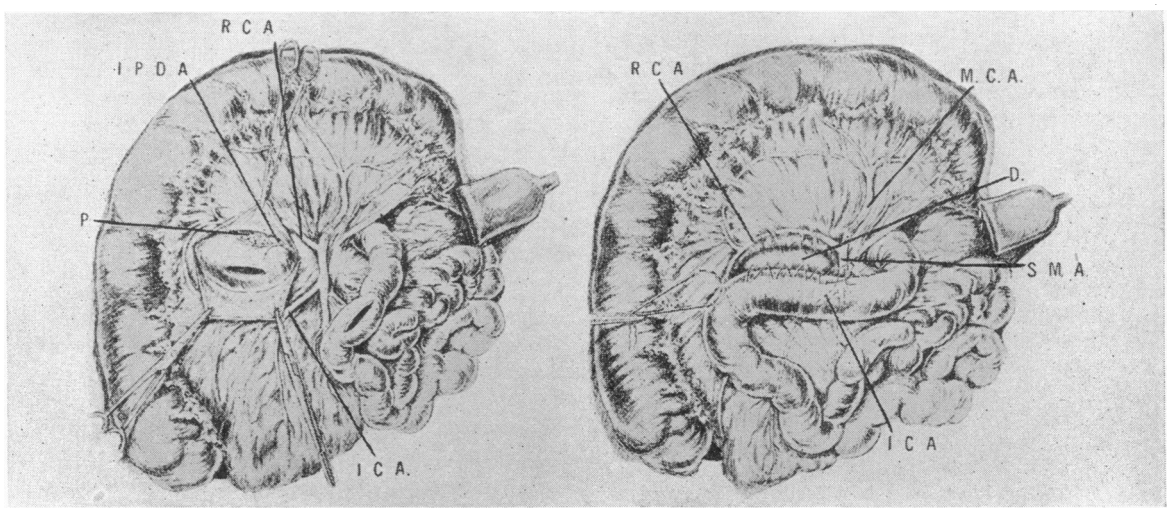

Fig. 3a Fig. 3b

Fig. 3a.—The duodenum is exposed between the right colic and ileocolic arteries. R.C.A., right colic artery; I.C.A., ileocolic artery; P., pancreas; I.P.D.A., inferior pancreatico-duodenal artery.

Fig. 3b.—An additional stitch is placed to prevent angulation. R.C.A., right colic artery; I.C.A., ileocolic artery; M.C.A., middle colic artery; D., dilated duodenum; S.M.A., superior mesenteric artery.

From J. McKenty: Chronic Duodenal Stenosis. (Permission for reproduction obtained from S. G. & O., and from author.)

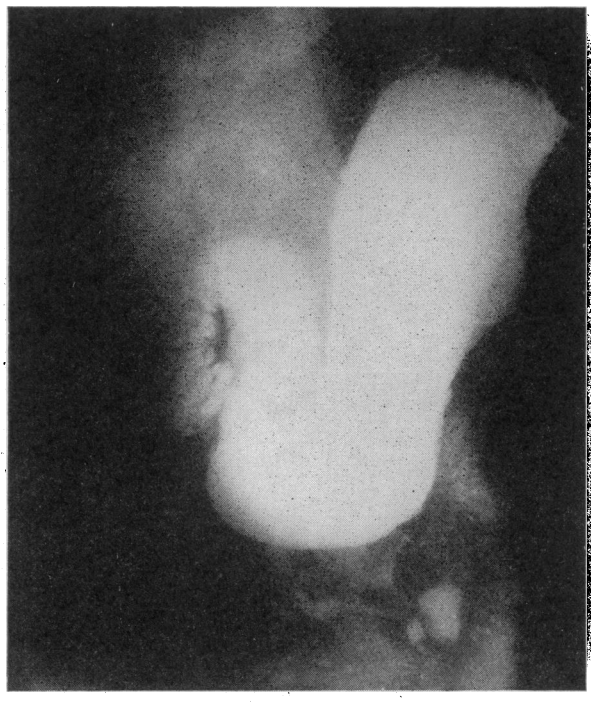

Fig. 4.—Roentgenogram, Case 1, showing chronic duodenal stasis.

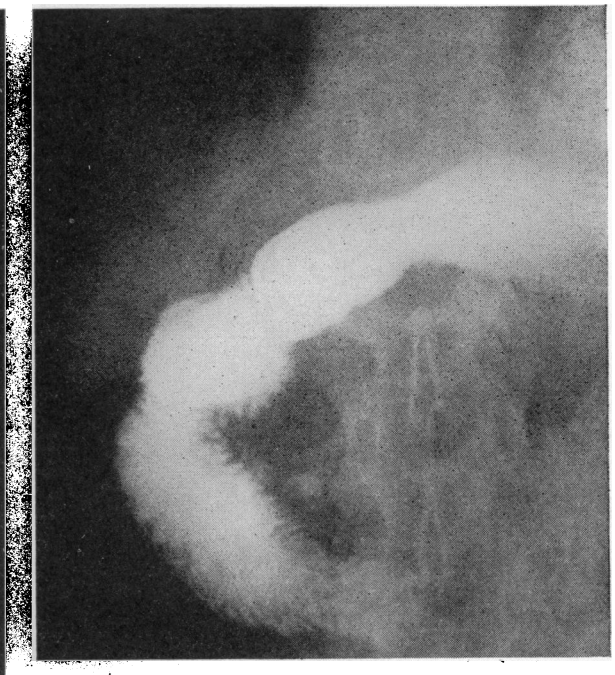

Fig. 5.—Roentgenogram, Case 2, chronic duodenal stasis. Duodenum visualized to the fourth portion.

or facial and cervical neuralgia, or even intracranial involvement.

I have abstracted the protocols of three illustrative cases which were referred because of neurological symptoms. A careful history in each case, a review of the physical and laboratory findings, and the confirmation by roentgenographic studies of the condition of chronic duodenal stasis led to the proper medical and postural treatment with relief of all symptoms, including the paramount neurological complaints.

REPORT OF CASES

CASE 1.—Mrs. L. A. M., A-277, age fifty-six, was admitted to the Stanford University Hospital on May 8, 1930, with the complaint of burning in the stomach for fourteen months, following an attack of influenza. The convalescence was prolonged with gastric distention, sour eructations, anorexia, nausea and vomiting, constipation, vertigo, tinnitus, severe headaches, and loss of fifty pounds in weight. Appendectomy a year ago brought no relief. Examinations of the patient, and of the urine and stools were negative. The Wassermann was negative. Her blood pressure was 125/85. Impression: possible migraine or gastric neurosis and psychoneurosis. Gastric analysis showed normal acid and low volume. Gastro-intestinal roentgenographic series showed chronic duodenal stasis (Fig. 4). The gall-bladder visualized normally. Treatment in the hospital consisted of frequent feedings, followed each time by the knee-chest position for fifteen minutes, and bed rest with the foot of the bed elevated. Gastric and neurological symptoms improved in three days, and the patient was discharged after a week of postural therapy with frequent feedings of selected bland diet. A belladonna and bromid mixture was prescribed and used in the hospital as well as at home, where bed rest was continued for a month. A report seven years later stated that she had experienced no return of symptoms, and had regained the fifty pounds in weight.

* * *

CASE 2.—Miss M. M., was seen by her physician in 1933, complaining of a bad headache, present for a period of years and, apparently, of a familial type. Two attacks of giant urticaria during the previous year were attributed to an allergic type of migraine. The headaches recurred every two to three weeks and were associated with nausea and vomiting. The day before the headache occurred she would be particularly hungry, and thirty hours after eating the headache would be intense, and she would vomit all the food she had taken. Thyroid extract was given for a low basal metabolic rate, but without relief, and the attacks became became more frequent and severe. Roentgenologically, an ulcer on the greater curvature was seen. However, on exploration, the stomach was normal, though the jejunum appeared to be pinched at the ligament of Treitz, and the constricting band was cut. Convalescence was uneventful, and the patient had no further trouble for a year, at which time the attacks recurred. I was asked to see her because of the severe headaches. Roentgenograms of her upper intestinal tract at this time revealed chronic duodenal stasis (Fig. 5). Relief was secured by dietary and postural measures.

* * *

CASE 3.—Miss E. M. K., A-59261, age sixteen, in the fall of 1936 complained of dizziness, headaches, vomiting, and some abdominal pain. A year before, a right radical mastoidectomy was followed by an uneventful convalescence of three weeks. A month later dizziness developed, and tinnitus appeared in the right ear. Nausea, with vomiting, occurred after meals, no matter how small the amount of food taken. Occasional colicky pains in the right lower quadrant were accompanied by tenderness in this region. Recently the mild bitemporal headaches became severe and bursting in character. Vestibular tests showed evidence of bilateral labyrinthitis, but there was no spontaneous nystagmus, nor other neurological signs. Impression: brain abscess and subacute appendicitis. However, these complaints in a thin, asthenic type of patient suggested chronic duodenal stasis, and this was confirmed roentgenologically (Fig. 6). The proper dietary and postural measures completely relieved the patient of all symptoms.

In four other patients the symptoms led to prompt roentgenological studies of the upper intestinal tract, which showed the characteristic picture of chronic duodenal stasis. Medical and postural measures gave successful results in each case.

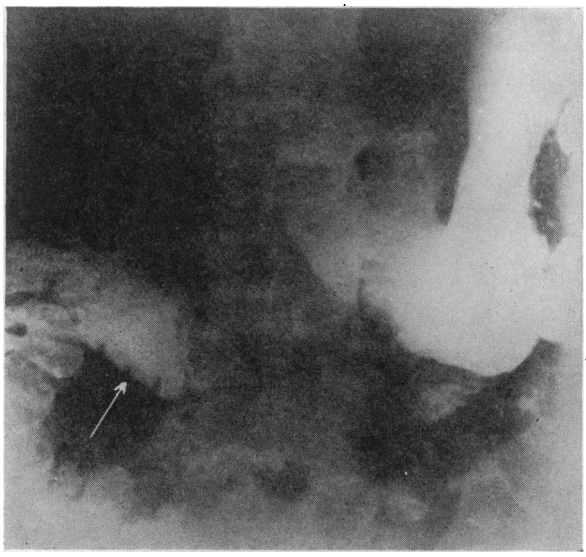

Fig. 6.—Roentgenogram, Case 3, chronic duodenal stasis. Visualization of the third portion of duodenum before treatment.

ETIOLOGY

In this paper consideration of the etiological factors will be limited to the form of chronic duodenal stasis resulting from pinching of the third portion of the duodenum between the root of the mesenteric vessels and the aorta. The same clinical features may be produced by congenital anomalies such as atresia, certain duodenal diverticula, annular pancreas, short mesentery and peritoneal bands. Another factor in its production may be the formation of adhesions such as those angulating the duodenum in certain cases of chronic cholecystitis, or chronic inflammatory thickening of the mesentery, or enlarged retroperitoneal or mesenteric glands, or inflammatory thickening of the ligament of Treitz, or the kinking of the duodenojejunal angle.

Spinal deformities, especially lordosis, low position of the duodenum, excessive motility of the colon, and the pelvic position of the intestines in visceroptosis, are also factors that favor compression of the duodenum.

In man, the third and fourth portions of the duodenum lie behind the peritoneal fold, and that portion behind the root of the mesentery has a flattened to ovoid form. Finney's [18] illustrations in 1906 emphasized the duodenal compression by the root of the mesentery in the erect posture, whereas the anatomical relations of these structures are best suited to the postures assumed by quadrupeds, as illustrated by Codman.[18] McKenty [2] felt that a loose cecum with an elongated parietocolic fold, having its support from the mesentery of the small bowel, is the important factor in the mesenteric occlusion of the duodenum.

SYMPTOMS

The stasis of duodenal and gastric secretions present a variety of symptoms, some of which are due to the toxicity of the duodenal contents. Indigestion and flatulency frequently are experienced one to three hours after meals. If the pylorus is tonic there may be pain, frequently burning in character, in the epigastrium; but if the pylorus is atonic and patulous, the duodenal contents regurgitate into the stomach, and vomiting, without pain, is a common symptom. The vomitus may be copious and, when it contains bile and pancreatic juices, suspicion of a dilated duodenum is aroused, especially in a visceroptotic patient.

Stavely,[17] in 1908, as well as subsequent authors, emphasized the lassitude, malaise, and particularly the headaches, usually frontal or temporal, frequently intense and dominant. Temporal, facial, cervical and scapular pains may be severe and suggest an atypical neuralgia. An associated vertigo may be present. The vomiting, headache, vertigo and other neurological symptoms, are suggestive of migraine or increased intracranial pressure.

After copious emesis, the so-called "bilious attacks" with their toxic symptoms are relieved. The patient quickly associates the taking of food with the onset of symptoms and fears to eat, thus producing a vicious circle, with ensuing loss of weight, anemia, and associated abdominal symptoms that may suggest cholecystitis or appendicitis. Constipation is the usual condition, but it may be accompanied by intermittent diarrhea when some of the toxic contents pass through into the lower intestines.

SIGNS

Examination reveals that most of the patients belong to the asthenic type with evidence of visceroptosis. Some show epigastric tenderness, slight secondary anemia, low blood pressure, and evidence of vasomotor instability.

Gastric analysis is not very helpful, but the roentgenological evidence is well defined and characteristic, permitting a definite diagnosis. Increased motility with duodenal peristalsis, and antiperistalsis and regurgitation of the barium into the stomach are seen by fluoroscopy. Four hours after the barium meal the dilated portions of the duodenum are usually visualized, particularly if the roentgenograms are taken with the patient in the upright position, front view and profile, then lying down, first in the ventral and then in the dorsal position.

TREATMENT

The majority of cases respond to medical therapy and postural treatment. In the case of acute dilatation of the stomach the repeated use of the stomach-tube is supplemented by postural treatment, including the prone position, the knee-chest position and the left lateral position, with the hips elevated. Likewise, in the treatment of chronic duodenal stasis dietary measures are supplemented by bed rest and the same postural treatment just mentioned.

Certainly, bed rest, with the foot of the bed elevated one to two feet, avoidance of the dorsal decubitus, and use of the lateral position, right or left, as frequently as possible, and the knee-chest position for fifteen or more minutes after meals, will overcome the stasis produced by compression of the duodenum by the root of the mesentery. Frequent feeding, every two to four hours, of a

bland, low residue diet, with high caloric and high vitamin value, will increase weight and, in time, deposit fat in the mesentery and improve the visceroptosis.

Abdominal exercises are instituted to strengthen the recti muscles. When the patient becomes ambulatory, a supporting corset or girdle is applied before arising, and worn when in the erect position to overcome the traction of the mesenteric pedicle and of the stomach.

Phenobarbital or bromids should be employed, and liquid petrolatum and agar used for constipation.

A small group of patients may not respond to such medical and postural measures. They usually show subsequent roentgenological evidence of duodenal stenosis, either from obstruction at the duodenojejunal angle, or duodenal fixation from adhesions, with gastric retention. In these patients conservative surgical measures will bring relief, such as division of constricting bands to the duodenum, or separation of adhesions of the mesentery in the pelvis, or suspension of the cecum and ascending colon or, where the cause of the mesenteric compression cannot be discovered, duodenojejunostomy. The postoperative care of these patients is important, with the employment of suitable diet, massage, postural treatment, and an abdominal belt during convalescence.

SUMMARY

The recognition of a clinical entity of chronic duodenal stasis thirty years ago was intimately associated with the recognition and treatment of acute dilatation of the stomach.

Compression of the third portion of the duodenum by traction on the mesentery containing the superior mesenteric vessels is a frequent cause of chronic duodenal stasis.

Symptoms of toxicity of the duodenal contents, lassitude, malaise, neuralgic pains, vertigo, and intense headaches may be paramount to the general symptoms of abdominal discomfort, indigestion, flatulency, vomiting, and constipation. When certain of these neurological symptoms are prominent, the picture may suggest migraine, atypical neuralgia, or even brain tumor.

Bilious attacks, with copious vomitus containing bile and pancreatic secretions, associated with headache and constipation, in an asthenic individual suggest chronic duodenal stasis.

The diagnosis is confirmed by roentgenological studies that reveal active peristalsis and antiperistalsis in a dilated duodenum.

The majority of cases will respond to medical and postural treatment, which include rest in a bed with its foot elevated one to two feet, frequent lateral and prone positions, the knee-chest posture for fifteen minutes after each meal, high caloric, high vitamin bland food taken every two to four hours, exercises to strengthen the abdominal wall, and the application of a supporting belt (which must be put on while the patient is in a recumbent position) when ambulatory.

Conservative surgical measures, as the last resort, include division of bands and adhesions, fixation of a mobile colon, or duodenojejunostomy.

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REFERENCES

1. Rokitansky, Carl: Lehrbuch der pathologischen anatomie. Ed. 3, 1863, III Wien.
2. Glenard, F.: De L'enteroptose, Presse Méd., 41:57-61, 1889.
3. Fagge, C. H.: On Acute Dilatation of the Stomach, Guy's Hosp. Rep., Series 3, 18:1-23, 1873.
4. Kussmaul, A.: Behandlung der Magenerweiterung durch eine neue Methode mittelst der Magenpumpe, Deutsches Arch. f. klin. Med., 6:455-501 (Dec.), 1869.
5. Schnitzler, J.: Ueber mesenteriale Darmincarceration, Wien. Klin. Rundschau, 9:579-593, 1895.
6. Kundrat: Ueber eine Seltene Form der inneren Inkarzeration, Abst. Wien. Med. Wchnschr., 41:351, 1891.
7. Albrecht, P. A.: Ueber arterio-mesenterialen Darmverschluss an der Duodeno-jejunalgrenze und seine ursächliche Beziehung zur Magenerweiterung, Virchow's Arch., 156:285-329, 1899.
8. Thomson, H. C.: Acute Dilatation of the Stomach, with Illustrative Cases, Lancet, 2:1113-1118, 1901, and Lancet, 2:287-289, 1902.
9. Laffer, W. B.: Acute Dilatation of the Stomach and Arterio-mesenteric Ileus, Ann. Surg., 47:390-417 and 532-558, 1908.
10. Dragstedt, L. R., Montgomery, M. L., Ellis, J. C., and Matthews, W. B.: The Pathogenesis of Acute Dilatation of the Stomach, Surg., Gynec. and Obst., 52:1057-1087 (June), 1931.
11. Zade, H.: Ueber postoperativen arterio-mesenterialen Darmverschluss an der Duodeno-jejunalgrenze und seinen Zusammenhang mit akuter Magendilatation, Beitr. z. Klin. Chir., 46:388-405, 1905.
12. Ochsner, A. J.: Constriction of the Duodenum Below the Entrance of the Common Duct and Its Relation to Disease, Trans. Am. S. A., 23:314-323, 1905.
13. Finney, J. M. T.: Gastro-mesenteric Ileus, Boston M. and S. J., 155:107-113, 1906.
14. Bloodgood, J. C.: Review of Abdominal Surgery, Internat. Clin., Vol. 1, Sixth Series, 1906.
15. Barker, L. F.: Discussion of J. M. T. Finney's paper on The Relation of Dilatation of the Duodenum to Gastric Disturbances, Johns Hopkins Hosp. Bull., 17:37 (Jan.), 1906.
16. Bloodgood, J. C.: Acute Dilatation of the Stomach, Gastromesenteric Ileus, Ann. Surg., 46:736-763, 1907.
17. Stavely, A. L.: Acute and Chronic Gastromesenteric Ileus, with Cure in a Chronic Case by Duodenojejunostomy, Johns Hopkins Hosp. Bull., 19:252-256, 1908.
18. Codman, E. A.: Chronic Obstruction of the Duodenum by the Root of the Mesentery, Boston M. and S. J., 158:503-511, 1908.
19. Wilkie, D. P. D.: Chronic Duodenal Ileus, Brit. M. J., 2:793-795, 1921.
20. McKenty, J.: Chronic Duodenal Stenosis, Surg. Gynec. and Obst., 38:444-450, 1924.
21. Higgins, C. C.: Chronic Duodenal Ileus, with Report of Fifty-Six Cases, Arch. Surg., 13:1-43, 1926.
22. Shattuck, H. F., and Imboden, H. M.: Chronic Intermittent Duodenal Obstruction, J. A. M. A., 98:943-947 (March 19), 1932.
23. Friedenwald, J., and Feldman, M.: The Unstable or Irritable Duodenum, J. A. M. A., 103:2007-2013 (Dec. 29), 1934.

Acknowledgment: Permission for reproduction of Fig. 2 and Fig. 3 was obtained from S. G. & O., and from the author, Dr. James McKenty.

DISCUSSION

NELSON J. HOWARD, M. D. (2957 Divisadero Street, San Francisco).—We are indebted to Doctor Reichert for his review of the anatomical and physiological factors producing occlusion of the third part of the duodenum through extraduodenal pressure. The symptom-complex of periodic severe headache, followed by copious vomiting usually of bile-stained material, deserves the reëmphasis given by Doctor Reichert. Awareness of this syndrome, and the

possibility of chronic duodenal stasis, will help the physician solve diagnostic difficulties, and with the conservative treatment outlined, the patients may be spared years of discomfort and ill health.

One personal experience:

Miss M. H. suffered for seven years from headache and insomnia. She was under treatment in the Stanford Clinic and had her teeth extracted, a nasal septum operation, glasses corrected, without relief. Laboratory investigation revealed no abnormal findings. Spinal puncture showed no increased pressure or pathological changes in the fluid. X-rays finally revealed what was interpreted as a large diverticulum of the second portion of the duodenum. By this time the patient had developed vomiting; severe, constant abdominal pain, and tenderness. Operation showed a duodenal stasis from encirclement of the duodenum by an annular pancreas.

Particularly is Doctor Reichert to be commended for showing us the effectiveness of simple conservative measures in most cases of chronic duodenal stasis.

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JOHN HOMER WOOLSEY, M.D. (Woodland Clinic, Woodland).—Chronic duodenal stasis is without doubt a clinical entity. It has been in the past recognized more often by a process of elimination than by direct diagnosis; but with the symptomatology better understood, it will become a condition more often thought of and, therefore, should be more promptly diagnosed. It is by the x-ray in competent hands that these clinical entities are being definitely demonstrated, and then proper treatment instituted. It follows, therefore, that the rule—that any patient with a gastro-intestinal complaint which does not respond to treatment under three weeks of proper and adequate diet and rest should have an adequate study of the gastro-intestinal tract by a competent roentgenologist—is most worth while.

As regards treatment, I am heartily in accord with conservative, or dietary and postural measures. Surgery should be employed only for the very severe or obdurate cases, and in those instances where a definite localized block more amenable to surgical correction has been demonstrated.

Duodenojejunostomy, performed according to the method without clamps, and with the mesocolon stitched well back on the duodenum, will, I believe, give a good result; but duodenojejunostomy as done, has not, from my observation, afforded the relief expected and desired.

EAR MANIFESTATIONS FOLLOWING HEAD INJURIES*

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DISCUSSION *by Harold A. Fletcher, M.D., San Francisco; Ben L. Bryant, M.D., Los Angeles; William J. Mellinger, M.D., Santa Barbara.*

INJURIES to the head are of interest to otologists, because many of them affect the ear mechanism in one or both of its divisions. It has been my observation that the severity of the injury often bears no relation to the development of cochlear or vestibular symptoms.

DIMINISHED HEARING

The most common ear symptom encountered following head injuries is a nerve deafness characterized by a decrease in hearing for high tones. The next symptom in order of frequency is labyrinthine vertigo, which is manifested by dizziness and aggravated by quick head movements.

Diminished hearing can easily be demonstrated by means of a careful hearing examination. The addition of the audiometer to our equipment has now greatly simplified this procedure, as well as given us a better record of hearing losses than we were able to obtain by the older methods. I might add also that a malingerer, claiming a hearing loss, may now be more easily detected by repeating this test several times. In a series of hundreds of audiometer tests repeated on coöperative patients, I have found that the readings will not vary more than 2 or 3 per cent.

LABYRINTHINE SYMPTOMS

The presence of labyrinthine symptoms may be demonstrated by quick head movements, or by the more accurate caloric and turning tests. Fortunately for the examiner in some insurance cases, the symptom of nystagmus is independent of the will and it is impossible for a malingerer to stimulate nystagmus.

In testing for labyrinthine symptoms the examiner must remember that these symptoms may be caused by other conditions than head injuries. He may encounter this symptom-complex in patients suffering from syphilis, also in patients with nicotin, alcohol, lead or carbon monoxid poisoning. Toxic labyrinthitis may develop secondary to a focal infection, and labyrinthine symptoms may appear as a manifestation of menopause. When any of these conditions are present, a hearing examination will often reveal the added symptom of nerve deafness.

THREE TYPES OF HEAD INJURIES

Three types of head injuries may produce a hearing loss and labyrinthine symptoms: First, concussion of the brain without skull fracture. Second, concussion of the brain with skull fracture, but not involving the petrous portion of the temporal bone. Third, concussion of the brain with skull fracture which involves the petrous portion of the temporal bone.

CONCUSSION OF THE BRAIN WITHOUT SKULL FRACTURE

Following a concussion of the brain, with or without skull fracture, we often see an increase in brain pressure. The brain substance, cerebrospinal fluid and blood vessels are all affected. The brain substance is compressed and may swell as a result of trauma. The blood vessels of the brain may become dilated and the circulation become sluggish because of a traumatic paralysis of the vasoconstrictor center. Exudate and lymphocytes may go out from the blood vessels into the brain substance. This circulation phenomena may persist after injury, and may be characterized by a concussion neurosis, namely, vasomotor instability, irregular pulse, and poor memory.

If the lateral ventricles are compressed, cerebrospinal fluid is forced into the third ventricle, then through the aqueduct of Sylvius into the fourth ventricle. The increased pressure on the floor of the fourth ventricle may affect the nuclei of the auditory nerve situated there. Degenerative changes may take place in these nuclei causing ear symptoms.

* Read before the Eye, Ear, Nose and Throat Section of the California Medical Association at the sixty-sixth annual session, Del Monte, May 2–6, 1937.